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### Tools and Links

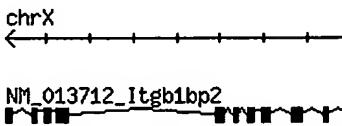
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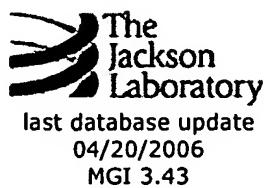
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Gene Detail		Your Input Welcom	
Symbol Name ID	<b>Itgb1bp2</b> integrin beta 1 binding protein 2 MGI:1353420	<a href="#">Nomenclature Hi:</a>	
Synonyms	Chordc3, melusin		
Genetic Map	Chromosome X cytoband D  Mapping data(2)		
Sequence Map	96050527-96055141 bp, + strand  (From NCBI annotation of NCBI Build 34)  <a href="#">UCSC Browser</a>   <a href="#">NCBI Map Viewer</a>	chrX 	<a href="#">MGI Mouse GBrowse</a>
Mammalian homology	human; rat <a href="#">(Mammalian Orthology)</a>		
Sequences	<p>Representative Sequences</p> <p><input type="checkbox"/> genomic 26549 <a href="#">NCBI Gene Model</a>   <a href="#">MGI Sequence Detail</a></p> <p><input type="checkbox"/> transcript NM_013712 <a href="#">RefSeq</a>   <a href="#">MGI Sequence Detail</a></p> <p><input type="checkbox"/> polypeptide Q9R000 <a href="#">SWISS-PROT</a>   <a href="#">EBI</a>   <a href="#">MGI Sequence Detail</a></p>	Length	Strain/Species F
		4615	C57BL/6J <input type="checkbox"/>
		1420	-
		350	Not Applicable
	<p>For the selected sequences <a href="#">download in FASTA format</a> <input type="checkbox"/> <a href="#">Go</a></p> <p>All sequences(14)</p>		
Phenotypes	<p>All phenotypic alleles(1) : Targeted, other(1)</p> <p>Mutant animals show normal cardiac structure and function under physiological conditions. When subjected to pressure overload, mutant hearts display contractile dysfunction and dilated cardiomyopathy.</p>		
Gene Ontology (GO) classifications	<p>Component <a href="#">Z disc</a>  Function <a href="#">calcium ion binding</a>, <a href="#">zinc ion binding</a>...  All GO classifications(3)</p>		
Expression	<p>GXD literature index(1) <a href="#">cDNA source data</a>(23)</p>		
Other database links	<p>DoTS <a href="#">DT.40176660</a>, <a href="#">DT.99748989</a>  UniGene <a href="#">46232</a>  TIGR <a href="#">TC1465282</a>, <a href="#">TC1555768</a>  NIA Mouse <a href="#">U020069</a></p>		



	Gene Index Entrez Gene <a href="#">26549</a>
Protein domains	InterPro ID Description <a href="#">IPR007051</a> CHORD <a href="#">IPR007052</a> CS <a href="#">IPR008978</a> HSP20-like chaperone <a href="#">Graphical View of Protein Domain Structure</a>
Molecular reagents	All nucleic(24) Genomic(1) cDNA(23)
References	(Earliest) <a href="#">J:57924</a> Brancaccio M <i>et al.</i> , "Melusin is a new muscle specific interactor for beta(1) integrin cytoplasmic domain." <i>J Biol Chem</i> 1999 Oct 8;274(41):29282-8 (Latest) <a href="#">J:93913</a> Kuninger D <i>et al.</i> , "Gene discovery by microarray: identification of novel genes induced during growth factor-mediated muscle cell survival and differentiation." <i>Genomics</i> 2004 Nov;84(5):876-89 <a href="#">All references(6)</a>



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## References

Query Results – Details

MGI Accession ID: MGI:2448192

J Number: J:81163

Other Accession IDs:

- 22402161 ([MEDLINE](#))
- 12496958 ([PubMed](#))

**Title:** Melusin, a muscle-specific integrin beta(1)-interacting protein, is required to prevent cardiac failure in response to chronic pressure overload.

**Authors:** Brancaccio M; Fratta L; Notte A; Hirsch E; Poulet R; Guazzone S; De Acetis M; Vecchione C; Marino G; Altruda F; Silengo L; Tarone G; Lembo G

**Journal:** Nat Med

**Volume:** 9

**Issue:** 1

**Date:** 2003 Jan

**Year:** 2003

**Pages:** 68-75

**Review Status:** Peer Reviewed

#### Abstract:

Cardiac hypertrophy is an adaptive response to a variety of mechanical and hormonal stimuli, and represents an early event in the clinical course leading to heart failure. By gene inactivation, we demonstrate here a crucial role of melusin, a muscle-specific protein that interacts with the integrin beta(1) cytoplasmic domain, in the hypertrophic response to mechanical overload. Melusin-null mice showed normal cardiac structure and function in physiological conditions, but when subjected to pressure overload—a condition that induces a hypertrophic response in wild-type controls—they developed an abnormal cardiac remodeling that evolved into dilated cardiomyopathy and contractile dysfunction. In contrast, the hypertrophic response was identical in wild-type and melusin-null mice after chronic administration of angiotensin II or phenylephrine at doses that do not increase blood pressure—that is, in the absence of cardiac biomechanical stress. Analysis of intracellular signaling events induced by pressure overload indicated that phosphorylation of glycogen synthase kinase-3beta (GSK-3beta) was specifically blunted in melusin-null hearts. Thus, melusin prevents cardiac dilation during chronic pressure overload by specifically sensing mechanical stress.

#### Additional Information:

- [Genes and Markers \(1\)](#)
- [Phenotypic Alleles \(1\)](#)

last database update  
04/20/2006  
MGI 3.43